Refine Search

Search Results -

Term	Documents
(2 NOT 3).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	15
(L2 NOT L3).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	15

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L4

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Tuesday, May 02, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=AND			
L4	L2 not L3	15	L4
L3	L2 and (tumor or cancer)	30	L3
L2	(IFNAR2c or huIFNAR2 or IFN-R)	45	L2
L1	Croze-Ed.in.	1	L1

END OF SEARCH HISTORY

PALM INTRANET

Day : Tuesday
Date: 5/2/2006
Time: 10:14:11

Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	**First Name**	
Croze	Ed	Search

To go back use Back button on your browser toolbar.

Back to PALM | ASSIGNMENT | OASIS | Home page

PALM INTRANET

Day : Tuesday
Date: 5/2/2006
Time: 10:14:11

Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	**First Name**	
Russell-Harde	Dean	Search

To go back use Back button on your browser toolbar.

Back to PALM | ASSIGNMENT | OASIS | Home page

PALM INTRANET

Day : Tuesday
Date: 5/2/2006
Time: 10:14:11

Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	**First Name**	
Vogel	David	Search

To go back use Back button on your browser toolbar.

Back to PALM | ASSIGNMENT | OASIS | Home page

```
                    Welcome to DialogClassic Web(tm)
 Dialog level 05.11.05D
Last logoff:  26apr06 13:18:28
Logon file001 02may06 09:34:19
          *** ANNOUNCEMENTS ***
                    ***
NEW FILES RELEASED
***Regulatory Affairs Journals (File 183)
***Index Chemicus (File 302)
***Inspec (File 202)

RESUMED UPDATING
***File 141, Reader's Guide Abstracts
                    ***
RELOADS COMPLETED
***File 516, D&B--Dun's Market Identifiers
***File 523, D&B European Dun's Market Identifiers
***File 531, American Business Directory
*** MEDLINE has been reloaded with the 2006 MeSH (Files 154 & 155)
*** The 2005 reload of the CLAIMS files (Files 340, 341, 942)
is now available online.

                    ***
Chemical Structure Searching now available in Prous Science Drug
Data Report (F452), Prous Science Drugs of the Future (F453),
IMS R&D Focus (F445/955), Pharmaprojects (F128/928), Beilstein Facts (F390), Derwent
(File 302).
                    ***
 >>>For the latest news about Dialog products, services, content<<<
 >>>and events, please visit What's New from Dialog at <<<
 >>>http://www.dialog.com/whatsnew/.  You can find news about<<<
 >>>a specific database by entering HELP NEWS <file number>.<<<
KWIC is set to 50.
HILIGHT set on as ' '
 * * *

File   1:ERIC 1966-2006/Mar (c) format only 2006 Dialog

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
?

B 155, 159, 5, 73
       02may06 09:34:32 User259876 Session D870.1
            $0.84    0.240 DialUnits File1
     $0.84  Estimated cost File1
     $0.05  INTERNET
     $0.89  Estimated cost this search
     $0.89  Estimated total session cost   0.240 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R) 1951-2006/May 03
         (c) format only 2006 Dialog
  File 159:Cancerlit 1975-2002/Oct
         (c) format only 2002 Dialog
 *File 159: Cancerlit is no longer updating.
Please see HELP NEWS159.
  File   5:Biosis Previews(R) 1969-2006/Apr W4
         (c) 2006 BIOSIS
```

```
  File  73:EMBASE 1974-2006/May 02
        (c) 2006 Elsevier Science B.V.

      Set  Items  Description
      ---  -----  -----------
?

S (IFNAR2C OR HUIFNAR2 OR IFN-R OR IFNR2)
             51  IFNAR2C
              3  HUIFNAR2
              2  IFN-R
              0  IFNR2
      S1      56  (IFNAR2C OR HUIFNAR2 OR IFN-R OR IFNR2)
?

S S1 AND (TUMOR OR CANCER)
             56  S1
        2699548  TUMOR
        2720139  CANCER
      S2      24  S1 AND (TUMOR OR CANCER)
?

RD
      S3      13  RD  (unique items)
?

S S3 NOT PY>2000
             13  S3
        8674046  PY>2000
      S4       4  S3 NOT PY>2000
?

T S4/3,K/ALL

  4/3,K/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

12911058   PMID: 11046044
 Receptor  for activated C-kinase (RACK-1), a WD motif-containing protein,
 specifically associates with the human type I IFN receptor.
  Croze E; Usacheva A; Asarnow D; Minshall R D; Perez H D; Colamonici O
  Department  of Immunology, Berlex Biosciences, Richmond CA 94804, USA. ed
croze@berlex.com
  Journal  of  immunology  (Baltimore,  Md. - 1950) (UNITED STATES)   Nov 1
2000,  165  (9)  p5127-32,  ISSN 0022-1767--Print  Journal Code: 2985117R
  Contract/Grant No.: CA55079; CA; NCI; GM54709; GM; NIGMS
  Publishing Model Print
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed

  The cytoplasmic domain of the human type I IFN receptor chain 2 ( IFNAR2c
 or  IFN-alphaRbetaL)  was  used  as  bait  in a yeast two-hybrid system to
identify novel proteins interacting with this region of the receptor. We...
  ...;  GE;  Repetitive  Sequences,  Amino  Acid--immunology--IM; Research
Support,  U.S.  Gov't,  P.H.S.;  Saccharomyces  cerevisiae--genetics--GE;
Tetradecanoylphorbol  Acetate--pharmacology--PD; Tryptophan; Tumor Cells,
Cultured; Two-Hybrid System Techniques
```

4/3,K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12804960 PMID: 10825167
Role of the intracellular domain of the human type I interferon receptor 2 chain (IFNAR2c) in interferon signaling. Expression of IFNAR2c **truncation mutants in U5A cells.**
Russell-Harde D; Wagner T C; Rani M R; Vogel D; Colamonici O; Ransohoff R M; Majchrzak B; Fish E; Perez H D; Croze E
Berlex Biosciences, Richmond, California 94804, the Cleveland Clinic Foundation, Cleveland, Ohio, 44195, USA.
Journal of biological chemistry (UNITED STATES) Aug 4 2000, 275 (31) p23981-5, ISSN 0021-9258--Print Journal Code: 2985121R
Contract/Grant No.: 2PO1 62220; PHS; CA55079; CA; NCI; GM54709; GM; NIGMS
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Role of the intracellular domain of the human type I interferon receptor 2 chain (IFNAR2c) in interferon signaling. Expression of IFNAR2c **truncation mutants in U5A cells.**
A human cell line (U5A) lacking the type I interferon (IFN) receptor chain 2 (**IFNAR2c**) was used to determine the role of the **IFNAR2c** cytoplasmic domain in regulating IFN-dependent STAT activation, interferon-stimulated gene factor 3 (ISGF3) and c-sis-inducible factor (SIF) complex formation, gene expression, and antiproliferative effects. A panel of U5A cells expressing truncation mutants of **IFNAR2c** on their cell surface were generated for study. Janus kinase (JAK) activation was detected in all mutant cell lines; however, STAT1 and STAT2 activation was observed only in U5A cells expressing full-length **IFNAR2c** and **IFNAR2c** truncated at residue 462 (R2.462). **IFNAR2c** mutants truncated at residues 417 (R2. 417) and 346 (R2.346) or **IFNAR2c** mutant lacking tyrosine residues in its cytoplasmic domain (R2.Y-F) render the receptor inactive. A similar pattern was observed for IFN-inducible STAT activation...

...ablated in U5A, R2.Y-F, R2.417, and R2.346 cell lines. The implications are that tyrosine phosphorylation and the 462-417 region of **IFNAR2c** are independently obligatory for receptor activation. In addition, the distal 53 amino acids of the intracellular domain of **IFNAR2c** are not required for IFN-receptor mediated STAT activation, ISFG3 or SIF complex formation, induction of gene expression, and inhibition of thymidine incorporation. These data demonstrate for the first time that both tyrosine phosphorylation and a specific domain of **IFNAR2c** are required in human cells for IFN-dependent coupling of JAK activation to STAT phosphorylation, gene induction, and antiproliferative effects. In addition, human and murine cells appear to require different regions of the cytoplasmic domain of **IFNAR2c** for regulation of IFN responses.
...; Research Support, U.S. Gov't, P.H.S.; STAT1 Transcription Factor; STAT2 Transcription Factor; Signal Transduction; Trans-Activators --metabolism--ME; Transcription Factors--metabolism--ME; **Tumor** Cells, Cultured; Viral Interference

4/3,K/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12314226 PMID: 10049744

Formation of a uniquely stable type I interferon receptor complex by interferon beta is dependent upon particular interactions between interferon beta and its receptor and independent of tyrosine phosphorylation.

Russell-Harde D; Wagner T C; Perez H D; Croze E

Department of Protein Biochemistry, Department of Immunology, Berlex Biosciences, Richmond, California 94804, USA.

Biochemical and biophysical research communications (UNITED STATES) Feb 16 1999, 255 (2) p539-44, ISSN 0006-291X--Print Journal Code: 0372516

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Human type I interferons (IFN) require two receptor chains, IFNAR1 and **IFNAR2c** for high affinity (pM) binding and biological activity. Our previous studies have shown that the ligand dependent assembly of the type I IFN receptor chains...

...for all type I IFNs. IFNbeta appears unique in its ability to assemble a stable complex of receptor chains, as demonstrated by the observation that **IFNAR2c** co-immunoprecipitates with IFNAR1 when cells are stimulated with IFNbeta but not with IFNalpha. The characteristics of such a receptor complex are not well defined...

... receptor assembly. To further characterize the factors required for formation of such a stable receptor complex we demonstrate using IFN stimulated Daudi cells that (1) **IFNAR2c** co-immunoprecipitates with IFNAR1 even when tyrosine phosphorylation of receptor chains is blocked with staurosporine, and (2) IFNbeta1b but not IFNalpha2, is present in the...

; Humans; Interferon Type I, Recombinant--metabolism--ME; Macromolecular Substances; Membrane Proteins; Models, Biological; Models, Molecular; Phosphorylation; **Tumor** Cells, Cultured

4/3,K/4 (Item 1 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

0013148312 BIOSIS NO.: 200100320151

The expression of interferon-alphareceptor 2C at diagnosis is associated with cytogenetic response in interferon-alpha-treated chronic myeloid leukemia patients

AUTHOR: Barthe Christophe (Reprint); Mahon Francois-Xavier (Reprint); Gharbi Marie-Josee (Reprint); Fabere Carole; Bilhou-Nabera Christelle (Reprint); Hochhaus Andreas; Reiffers Josy (Reprint); Marit Gerald (Reprint)

AUTHOR ADDRESS: Hematology, University, Bordeaux, France**France

JOURNAL: Blood 96 (11 Part 1): p738a November 16, 2000 2000

MEDIUM: print

CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201

SPONSOR: American Society of Hematology

ISSN: 0006-4971

DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster

RECORD TYPE: Abstract

LANGUAGE: English

...ABSTRACT: to whom an alternative therapy may be proposed. In this study, the levels of expression of both BCR-ABL and subunit 2c of IFNa receptor (**IFNaR2c**) genes were analyzed at diagnosis in 74 chronic phase CML patients treated with an IFNa monotherapy. By using blood samples, real-time quantitative PCR (LightCycler technology) was performed to quantify BCR-ABL, **IFNaR2c** and G6PDH mRNA as an external control. The results were compared with hematological and cytogenetic response to IFNa. A wide variation of BCR-ABL/G6PDH...

...range 0.18 -41.3), but no significant association with either response to IFNa or other prognostic factors was observed. In contrast, the variation of **IFNaR2c** /G6PDH ratio at diagnosis was significantly associated with the achievement of major cytogenetic response (MCR ; < 34% Ph+ metaphases). Median values of **IFNaR2c** /G6PDH ratio for patients achieving MCR and for those who did not achieve it were 110.8% (range 9 - 612) and 64.4% (range 6...

...value), the probabilities to be in MCR at 24 months was 75 +- 19% but was 40% +-17% for the other group i.e.patients with **IFNaR2c** /G6PDH ratio < 78.8% (p = 0.024). In addition, this novel independent molecular factor combined with the achievement of complete hematological response at three months...

...90.4% +- 18% at 24 months; p = 0.00001). So, in the current study, we show for the first time that the expression level of **IFNaR2c** mRNA is variable at diagnosis in CML patients and is statistically associated with IFNa response.

DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ...diagnostic **tumor** expression, drug-induced cytogenetic **tumor** response association

?

```
Set     Items   Description
S1         56   (IFNAR2C OR HUIFNAR2 OR IFN-R OR IFNR2)
S2         24   S1 AND (TUMOR OR CANCER)
S3         13   RD  (unique items)
S4          4   S3 NOT PY>2000
?

S S3 NOT S4
            13  S3
             4  S4
      S5     9  S3 NOT S4
?

T S5/3,K/ALL
```

5/3,K/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

14923527 PMID: 15185340

Interferon receptor expression regulates the antiproliferative effects of interferons on cancer cells and solid tumors.

Wagner T Charis; Velichko Sharlene; Chesney Steven K; Biroc Sandra; Harde Dean; Vogel David; Croze Ed

Department of Immunology, Berlex Bioscience Inc., Richmond, CA 94804, USA.

International journal of cancer. Journal international du cancer (United States) Aug 10 2004, 111 (1) p32-42, ISSN 0020-7136--Print
Journal Code: 0042124
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Interferon receptor expression regulates the antiproliferative effects of interferons on cancer cells and solid tumors.

... potent antiproliferative and immunomodulatory activities. Because of these properties IFNs have been evaluated as therapeutics for the treatment of a number of human diseases, including **cancer** . Currently, IFNs have been shown to be efficacious for the treatment of only a select number of cancers. The reason for this is unclear. Recent evidence has demonstrated that some **cancer** cell types seem to be defective in their ability to respond to IFN. It has been suggested that defects in IFN signaling is one mechanism by which **cancer** cells escape responsiveness to Type I IFNs and growth control in general. We report that transfection and enhanced expression of the Type I IFN receptor chain (**IFNAR2c**) in 3 different human **cancer** cell lines markedly increases the sensitivity of these cells to the antiproliferative effects of IFNs. In **cancer** cells transfected with **IFNAR2c** , dose response curves demonstrate a significant decrease in the concentrations of IFN required to achieve maximum cell death. Furthermore, in these transfected cells, we observe...

... significant increase in the number of cells undergoing apoptosis, as measured by DNA fragmentation and Caspase 3 activation. In addition, using an in vivo xenograft **tumor** model we show an increase in the effectiveness of systemically delivered Betaseron in decreasing **tumor** burden in animals in which solid tumors were generated from **IFNAR2c** transfected cells. These data show that specific regulation of IFN receptor expression can play a major role in determining the clinical outcome of IFN-based **cancer** therapeutics by regulating the relative sensitivity of **cancer** cells to IFN-dependent growth control. Copyright 2004 Wiley-Liss, Inc.
; Animals; Apoptosis; Caspases--pharmacology--PD; Cell Line, **Tumor** ; DNA Damage; Gene Therapy--methods--MT; Humans; Mice; Mice, Nude; Receptors, Interferon--physiology--PH; Research Support, Non-U.S. Gov't; Signal Transduction; Transfection; Transplantation...

5/3,K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13924581 PMID: 12105218

STAT3 activation by type I interferons is dependent on specific tyrosines located in the cytoplasmic domain of interferon receptor chain 2c. Activation of multiple STATS proceeds through the redundant usage of two tyrosine residues.

Velichko Sharlene; Wagner T Charis; Turkson James; Jove Richard; Croze Ed
Department of Immunology, Berlex Biosciences Inc., Richmond, California 94804 and the Molecular Oncology and Drug Discovery Programs, H. Lee Moffitt Cancer Center and Research Institute, Tampa, Florida 33612.

Journal of biological chemistry (United States) Sep 20 2002, 277 (38) p35635-41, ISSN 0021-9258--Print Journal Code: 2985121R
Publishing Model Print-Electronic
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: MEDLINE; Completed

... remains unclear. Understanding the IFN-dependent regulation of STAT3 is of increasing interest because recent studies have demonstrated that STAT3 may play a role in **cancer** . Studies have revealed that STAT3 is constitutively active in a number of **cancer** cell lines and that overexpression of an active form of STAT3 transforms normal fibroblasts. Therefore, STAT3 exhibits properties indicative of known oncogenes. In this report...

...the role of the type I IFN receptor in STAT3 activation and identify for the first time tyrosine residues present in the cytoplasmic domain of **IFNAR2c** that are critical for STAT3 activation. The regulation of STAT3 activation by IFNs was measured in a human lung fibrosarcoma cell line lacking **IFNAR2c** but stably expressing various **IFNAR2c** tyrosine mutants. We show here that in addition to IFN-dependent tyrosine phosphorylation of STAT3, activation using a STAT3-dependent electrophoretic mobility shift assay and...

... type I IFN-dependent activation of STAT3 proceeds through a novel mechanism that is dependent on two tyrosines, Tyr(337) and Tyr(512), present in **IFNAR2c** and contained within a conserved six-amino acid residue motif, GxGYxM. Surprisingly, both tyrosines were previously shown to be required for type I IFN-dependent...

... activation. Our results reveal that type I IFNs activate multiple STATs via the overlapping usage of two tyrosine residues located in the cytoplasmic domain of **IFNAR2c**.
; Base Sequence; DNA Primers; Electrophoretic Mobility Shift Assay; Humans; Membrane Proteins; Receptors, Interferon--chemistry--CH; STAT3 Transcription Factor; **Tumor** Cells, Cultured

5/3,K/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13673903 PMID: 11910354
Interferon-alpha activates multiple STAT signals and down-regulates c-Met in primary human hepatocytes.
Radaeva Svetlana; Jaruga Barbara; Hong Feng; Kim Won-Ho; Fan Saijun; Cai Hongbo; Strom Stephen; Liu Youhua; El-Assal Osama; Gao Bin
Section on Liver Biology, Laboratory of Physiologic Studies, National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health, Bethesda, Maryland 20892, USA.
Gastroenterology (United States) Apr 2002, 122 (4) p1020-34, ISSN 0016-5085--Print Journal Code: 0374630
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

... The differential response to IFN-alpha stimulation in primary human and mouse hepatocytes may be caused by expression of predominant functional IFN-alpha receptor 2c (**IFNAR2c**) in primary human hepatocytes vs. expression of predominant inhibitory IFNAR2a in mouse hepatocytes. Microarray analyses of primary human hepatocytes show that IFN-alpha up-regulates about 44 genes by over 2-fold and down-regulates about 9 genes

by 50%. The up-regulated genes include a variety of antiviral and **tumor** suppressors/proapoptotic genes. The down-regulated genes include c-myc and c-Met, the hepatocyte growth factor (HGF) receptor. Down-regulation of c-Met is...
...; Rats, Sprague-Dawley; Receptors, Interferon--genetics--GE; STAT1 Transcription Factor; STAT2 Transcription Factor; STAT3 Transcription Factor; STAT5 Transcription Factor; Solubility; Sp1 Transcription Factor --metabolism--ME; **Tumor** Cells, Cultured; Up-Regulation--drug effects--DE

5/3,K/4 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2006 BIOSIS. All rts. reserv.

0015482626 BIOSIS NO.: 200510177126
T cell responses to MICA
AUTHOR: Zhang Y (Reprint); Stastny P
AUTHOR ADDRESS: Univ Texas, SW Med Ctr, Dallas, TX USA**USA
JOURNAL: Human Immunology 65 (Suppl. 1): pS14 2004 2004
CONFERENCE/MEETING: 30th Annual Meeting of the American-Society-for-Histocompatibility-and-Immunogenetics San Antonio, TX, USA October 02 -06, 2004; 20041002
SPONSOR: Amer Soc Histocompatibil & Immunogenet
ISSN: 0198-8859
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ...TNF-alpha (**tumor** necrosis factor-alpha
...

... **IFN-r**

5/3,K/5 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2006 BIOSIS. All rts. reserv.

0014746186 BIOSIS NO.: 200400116943
The differences of the signaling and response to type I interferons in hepatocellular carcinoma cell lines.
AUTHOR: Damdinsuren Bazarragchaa (Reprint); Nagano Hiroaki (Reprint); Sakon Masato (Reprint); Yamamoto Tameyoshi (Reprint); Ota Hideo (Reprint); Nakamura Masato (Reprint); Marubashi Shigeru (Reprint); Miyamoto Atsushi (Reprint); Umeshita Koji (Reprint); Dono Keizo (Reprint); Nakamori Shoji (Reprint); Monden Morito (Reprint)
AUTHOR ADDRESS: Graduate School of Medicine, Osaka University, Suita, Osaka, Japan**Japan
JOURNAL: Hepatology 38 (4 Suppl. 1): p408A-409A October 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 54th Annual Meeting of the American Association for the Study of Liver Diseases Boston, MA, USA October 24-28, 2003; 20031024
SPONSOR: American Association for the Study of Liver Diseases
ISSN: 0270-9139 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: gene products that determine the responses. However general

pathways of IFN signaling is established, the roles of individual signaling components and IFNARs in IFNs' anti- **tumor** effect against HCC are not yet been clarified. Moreover, the specific responses in signaling to type I IFNs (-alpha and -beta) have not been examined...

...diverse response of the cells may causes by expression differences of IFNARs and IFN signal transference proteins. By Western blot analysis the expressions of functional - **IFNAR2c** subunit (long form) and STAT1, 3 proteins were higher in IFN sensitive - PLC/PRF/5 cells than in resistant - HuH7 cell line. Alternatively, the expressions...

...IFNs, the tyrosine phosphorylations of STAT1 and STAT3, but not of STAT2, were greater in PLC/PRF/5 compared with HuH7 cells. Consequently those of **IFNAR2c** subunit and STAT1, 3 proteins may correlate with the IFN sensitivity of HCC. On the other hand, growth-inhibitory effect of IFN-beta was significantly...

DESCRIPTORS:
...MAJOR CONCEPTS: **Tumor** Biology
CHEMICALS & BIOCHEMICALS: ... **IFNAR2c** ;

5/3,K/6 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014106526 BIOSIS NO.: 200300065245
Multiple STAT activation by type I interferons is mediated by common tyrosine residues located in the cytoplasmic domain of IFNAR2c.
AUTHOR: Velichko Sharlene (Reprint); Wagner T Charis (Reprint); Vogel David (Reprint); Turkson James; Jove Richard; Croze Ed (Reprint)
AUTHOR ADDRESS: Immunology, Berlex Bioscience, Richmond, CA, 94804, USA** USA
JOURNAL: Journal of Interferon and Cytokine Research 22 (Supplement 1): p S-91 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Joint Meeting of the International Society for Interferon and Cytokine Research, the International Cytokine Society, the Society for Leukocyte Biology, and the European Cytokine Society on Cytokines and Interferons Turin, Italy October 06-10, 2002; 20021006
SPONSOR: International Society for Interferon and Cytokine Research
ISSN: 1079-9907 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

Multiple STAT activation by type I interferons is mediated by common tyrosine residues located in the cytoplasmic domain of IFNAR2c.
DESCRIPTORS:
DISEASES: **cancer** --
CHEMICALS & BIOCHEMICALS: ... **IFNAR2c** --

5/3,K/7 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014106470 BIOSIS NO.: 200300065189
Enhanced expression of the interferon receptor, IFNAR2c **, sensitizes both** cancer **cells and solid tumors to the antiproliferation effects of type I Interferons.**

AUTHOR: Wagner T Charis (Reprint); Chesney Steven K (Reprint); Velichko Sharlene (Reprint); Biroc Sandra (Reprint); Harde Dean (Reprint); Vogel David (Reprint); Croze Ed (Reprint)
AUTHOR ADDRESS: Departments of Immunology and Animal Pharmacology, Berlex Biosciences Inc., Richmond, CA, 94804, USA**USA
JOURNAL: Journal of Interferon and Cytokine Research 22 (Supplement 1): p S-73 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Joint Meeting of the International Society for Interferon and Cytokine Research, the International Cytokine Society, the Society for Leukocyte Biology, and the European Cytokine Society on Cytokines and Interferons Turin, Italy October 06-10, 2002; 20021006
SPONSOR: International Society for Interferon and Cytokine Research
ISSN: 1079-9907 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

Enhanced expression of the interferon receptor, IFNAR2c , **sensitizes both** cancer **cells and solid tumors to the antiproliferation effects of type I Interferons.**

DESCRIPTORS:
...MAJOR CONCEPTS: **Tumor** Biology
...DISEASES: **cancer** --
CHEMICALS & BIOCHEMICALS: ... **IFNAR2c** --
MISCELLANEOUS TERMS: ... **tumor** prevention...

... **tumor** volume

5/3,K/8 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013777336 BIOSIS NO.: 200200370847

STAT3 activation by type I interferons is mediated by specific tyrosines located in the cytoplasmic domain of the interferon receptor chain IFNAR2c

AUTHOR: Velichko Sharlene (Reprint); Wagner T Charis (Reprint); Vogel David (Reprint); Turkson James; Jove Richard; Croze Ed (Reprint)
AUTHOR ADDRESS: Immunology, Berlex Biosciences, 15049 San Pablo Avenue, Richmond, CA, 94804-0099, USA**USA
JOURNAL: FASEB Journal 16 (5): pA1222 March 22, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of Professional Research Scientists on Experimental Biology New Orleans, Louisiana, USA April 20-24, 2002; 20020420
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

STAT3 activation by type I interferons is mediated by specific tyrosines located in the cytoplasmic domain of the interferon receptor chain IFNAR2c

...ABSTRACT: roles in apoptosis. This observation places IFNs on a list of cellular modifiers that are able to regulate processes of transformation and malignant progression in **cancer** . The role of STAT1 and STAT2 in IFN signaling is well established; however, the mechanism of activation of STAT3 is unclear. The regulation of STAT3 by IFN has become increasingly

important in light of recent results demonstrating oncogene-like constitutive activation of STAT3 in **cancer** cells. In this report we identify for the first time a mechanism of STAT3 activation occurring via the redundant usage of two single tyrosines present in **IFNAR2c** . STAT3 activation is measured in a human **cancer** cell line (U5A) stably expressing a number of **IFNAR2c** tyrosine mutants. IFN-dependent transcriptional factor formation (STAT3:STAT3) and STAT3 specific reporter activation are also described. In addition, it is shown that STAT3 activation...
DESCRIPTORS:
MAJOR CONCEPTS: **Tumor** Biology
...ORGANISMS: human **cancer** cell line
CHEMICALS & BIOCHEMICALS: ... **tumor** cell cytoplasmic domain tyrosines, **tumor** cell expression, type I interferon-induced STAT-3 protein activation mediator...

... **tumor** cell expression, type Interferon activation

5/3,K/9 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2006 Elsevier Science B.V. All rts. reserv.

12750176 EMBASE No: 2004344184
Initial expression of interferon alpha receptor 2 (IFNAR2) on CD34-positive cells and its down-regulation correlate with clinical response to interferon therapy in chronic myelogenous leukemia
Ito K.; Tanaka H.; Ito T.; Sultana T.A.; Kyo T.; Imanaka F.; Ohmoto Y.; Kimura A.
Dr. H. Tanaka, Dept. of Hematology and Oncology, Res. Inst. Radiat. Biol. and Med., Hiroshima University, 1-2-3 Kasumi, Minami-ku, Hiroshima 734-8553 Japan
AUTHOR EMAIL: dtanaka@hiroshima-u.ac.jp
European Journal of Haematology (EUR. J. HAEMATOL.) (United Kingdom) 2004, 73/3 (191-205)
CODEN: EJHAE ISSN: 0902-4441
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 52

...of IFNAR2 expression during IFNalpha therapy was observed only in good responders but not in poor responders. In addition to protein level, both initial high **IFNAR2c** mRNA expression level and its down-regulation during IFNalpha therapy, in purified CD34-positive cells, were also observed only in good responders. In contrast to...
MEDICAL DESCRIPTORS:
*receptor down regulation; * **cancer** immunotherapy; *chronic myeloid leukemia--drug therapy--dt
SECTION HEADINGS:
016 **Cancer**
025 Hematology
026 Immunology, Serology and Transplantation
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
?

Set	Items	Description
S1	56	(IFNAR2C OR HUIFNAR2 OR IFN-R OR IFNR2)
S2	24	S1 AND (TUMOR OR CANCER)

```
S3          13   RD  (unique items)
S4           4   S3 NOT PY>2000
S5           9   S3 NOT S4
?

COST
       02may06 09:41:57 User259876 Session D870.2
            $1.40    0.412 DialUnits File155
               $1.32  6 Type(s) in Format  3
            $1.32  6 Types
     $2.72  Estimated cost File155
            $0.44    0.139 DialUnits File159
     $0.44  Estimated cost File159
            $2.48    0.420 DialUnits File5
               $0.96  6 Type(s) in Format 95 (KWIC)
            $0.96  6 Types
     $3.44  Estimated cost File5
            $4.18    0.373 DialUnits File73
               $3.10  1 Type(s) in Format  3
            $3.10  1 Types
     $7.28  Estimated cost File73
            OneSearch, 4 files,  1.344 DialUnits FileOS
     $2.13  INTERNET
    $16.01  Estimated cost this search
    $16.90  Estimated total session cost   1.583 DialUnits
?
```

Return to logon page!